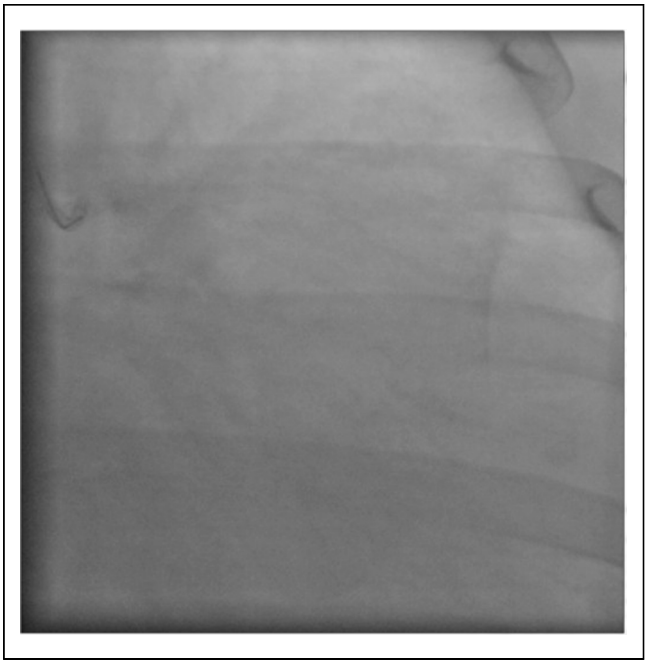
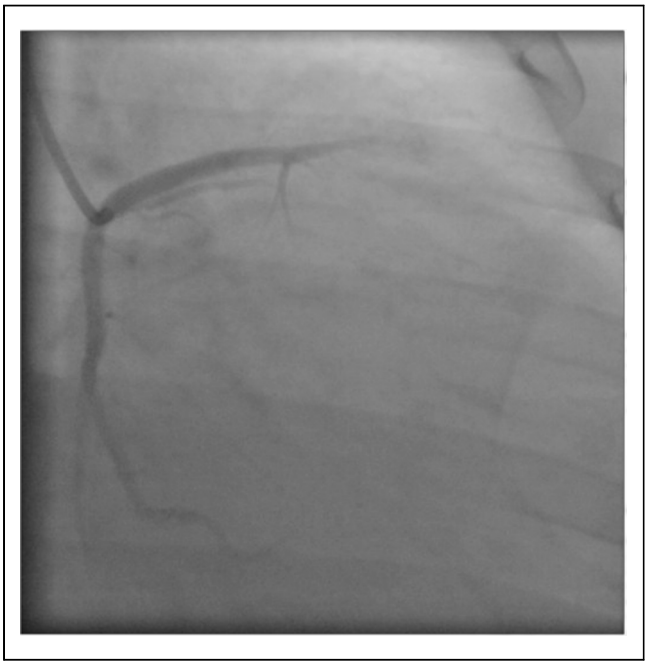
abrupt pulmonary edema was occurred after cardiac rehabilitation for 4 weeks.

After the control of pulmonary edema, second CAG was performed. LAD flow was jeopardized because of sub-acute thrombosis. Lumen narrowing was seen in the proximal LAD. TLR was performed with long DES to cover the lesion fully.





Case Summary. We experienced a young female case of acute myocardial infarction with a quantity of thrombus derived from multiple ruptured plaques with thin-cap fibro atheroma, which advanced to sub-acute thrombosis with abrupt pulmonary edema.

TCTAP C-016

"Single" Vessel, "Double" Primary Angioplasty, "Triple" Nightmares

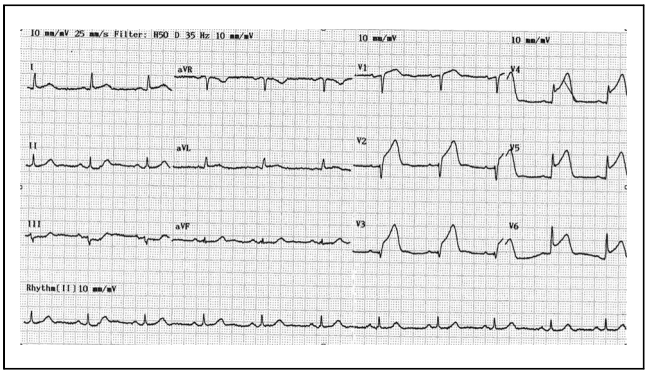
Kevin Kwok[1]
[1]Queen Elizabeth Hospital, Hong Kong, China

[CLINICAL INFORMATION]

Patient initials or identifier number. YNL

Relevant clinical history and physical exam. YNL is a 66 years old chronic smoker who enjoyed well past health. He was admitted for sudden onset severe chest pain without hemodynamic compromise. Physical examination showed normal JVP, clear chest and dual heart sound without significant heart murmur.

Relevant test results prior to catheterization. Electrocardiogram on admission showed ST segment elevation over anterior precordial leads. Echocardiogram showed impaired left ventricular systolic function with hypokinetic myocardium supplied by left anterior descending artery. CXR showed normal mediastinum without evidence of pulmonary edema.



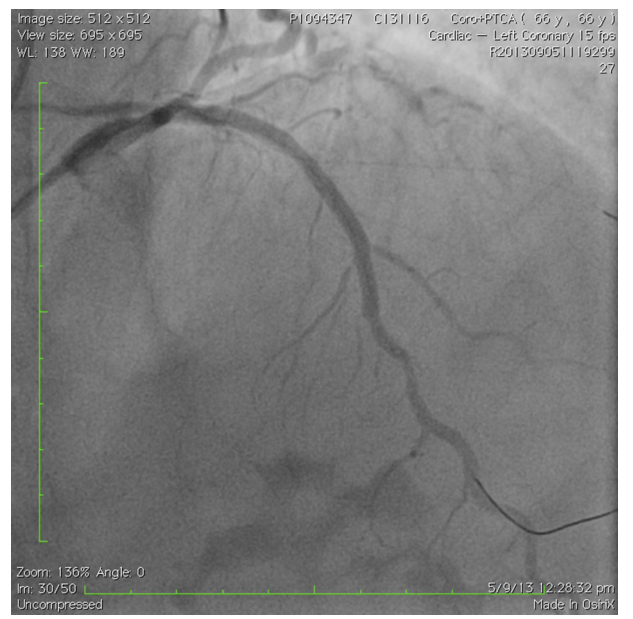
Relevant catheterization findings. Urgent coronary angiogram confirmed acute proximal left anterior descending artery thrombotic occlusion. Left circumflex artery and right coronary artery were unremarkable.

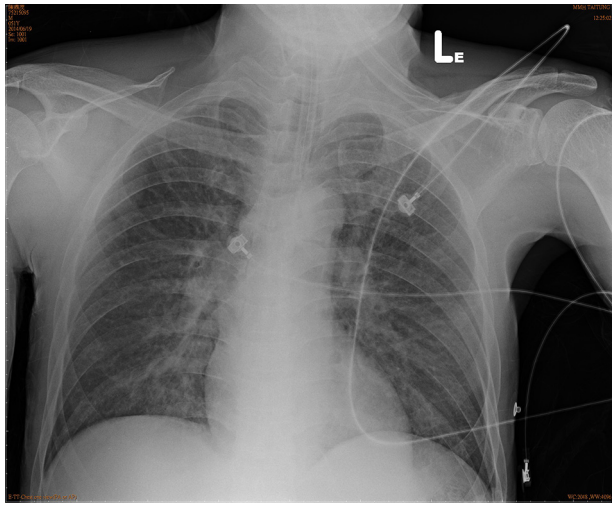
[INTERVENTIONAL MANAGEMENT]

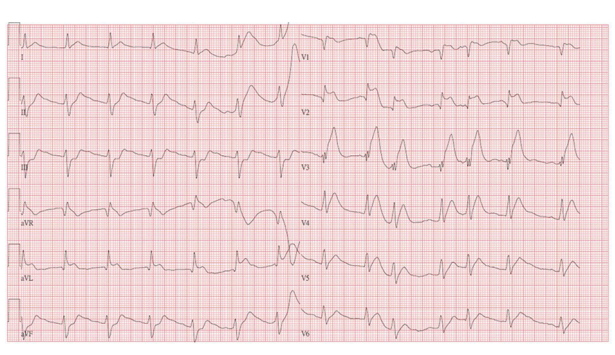
Procedural step. Right radial approach with 6Fr IL3.5 guiding catheter was used. Thrombotic occlusion was crossed with Runthrough guidewire and GP2b3a inhibitor was given. Thrombectomy device aspirated small amount of white thrombus. An underlying critical stenosis at LAD/ D1 bifurcation (medina 1,1,1 lesion) was found. D1 was also wired and bifurcation lesions were sequentially pre-dilated. Proximal to mid LAD lesions were then stented with 3.0 x 22mm and 3.0 x 26mm DES with overlapping. D1 was then found jailed after LAD stenting.

Suddenly patient developed severe chest pain with cardiogenic shock while attempting to rewire D1 acute closure. Extensive thrombus was found in LMN extending to LAD and LCx. Hemodynamic was immediately supported by high dose inotropes and IABP. However patient then developed recurrent malignant arrhythmia and acute pulmonary edema, which necessitated multiple defibrillation/ cardio version and mechanical ventilation. After repeated cardiopulmonary resuscitation and severe patient agitation despite sedation, guiding catheter and all guidewires were totally lost.

Femoral approach with 7Fr EBU3.5 guiding was then switched for better support. LAD and LCx were carefully re-wired. LCx was reopened first and LMN-LAD was then stented with 4.0 x 22 with overlapping to LAD stent. Final kissing inflation to LAD/LCx was performed. Final angiogram showed TIMI 3 flow. Patient finally survived and was successfully discharged from hospital after intensive support.







Case Summary. We presented a complicated case when acute LAD thrombotic occlusion first became bifurcation lesion with D1 occlusion, then LMN occlusion with circulatory collapse and finally guiding catheter/guidewire lost in a dying heart. However we managed to save the patient with satisfactory outcome.

Highlighted discussion

Choose stenting strategy (simple vs complex) in case of difficult bifurcation lesion in primary angioplasty setting.

TCTAP C-017

STEMI Caused by Left Main Trunk Total Occlusion and Rescued by Primary PCI+TPM+IABP+PCPS

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[CLINICAL INFORMATION]

Patient initials or identifier number. hospital ID number: 75215095

Relevant clinical history and physical exam. A 51-year-old man was sent to the emergency department with a complaint of typical retrosternal chest pain lasting for 1 hours accompanied by cold sweating. He had been smoking 0.5PPD for 30 years and a history of hypertension for years with medication control. The patient had no history of angina pectoris before and no family history of CAD diseases. In the emergency department, he lost his consciousness and developed VT/VF requiring direct-current cardio version, CPR and emergency intubation.

Relevant test results prior to catheterization. On physical examination, his blood pressure couldn't be measured, and his pulse rate was 76 beats/min and regular. The ECG showed atrial fibrillation, left axis deviation and ST-segment elevation in leads aVR, I, aVL, and V1-V6. The plasma levels of both troponin I and creatinine kinase-MB on emergency department were normal: <0.05 ng/mL(reference value, <0.4 ng/mL) and <1 ng/mL, but myoglobin was high: 379ng/ml, respectively. The other laboratory findings were as below.

Lab Data

(Hemoglobin)	13.6	g/dL
(Hematocrite)	42.0	%
(W.B.C)	22.60	10^3/uL
(WBC differential count)		
(Segmented neutrophil)	56.0	%
(Eosinophil)	0.0	%
(Baso)	0.0	%
(Monocyte)	8.0	%
(Lymphocyte)	36.0	%
(Platelet)	245	10^3/uL

(Glucuse AC)	835(Recheck)	mg/dL
(S-GOT)	120	IU/L
(CK)	44	IU/L
(BUN)	22	mg/dL
(Creatinine)	1.5	mg/dL
(K)	3.1	mEq/L
(Na)	136	mEq/L
CKMB	<1.00	ng/mL
Myoglobin	379.0	ng/mL
Troponin-I	<0.05	ng/ml

Relevant catheterization findings. The coronary angiogram revealed a significant thrombus-containing total occlusion in the left main